AN 1993:24009176 BIOTECHNO
TI Actions of interleukin-4 on prostaglandin biosynthesis at the chorion-decidual interface
AU Adamson S.; Edwin S.S.; LaMarche S.; Mitchell M.D.
CS Department of Obstetrics/Gynecology, Univ. of Utah School of Medicine, 50 North Medical Dr.,Salt Lake City, UT 84132, United States.
SO American Journal of Obstetrics and Gynecology, (1993), 169/6 (1442-1447)
CODEN: AJOGAH ISSN: 0002-9378
DT Journal; Article
CY United States
LA English
SL English
AB Objective: We determined the effects of interleukin-4 on chorion and decidual prostaglandin production. Study design: Chorion and decidual cells from term placentas were grown to confluence. Cells were then incubated with interleukin-4 either alone or with other known stimulants of prostaglandin production: interleukin-1β, epidermal growth factor, ionomycin, or phorbol 12-myristate 13-acetate. Prostaglandin E.sub.2 production was determined with a specific radioimmunoassay. Results: Interleukin-4 alone stimulated prostaglandin E.sub.2 production in chorion and decidual cells. Interleukin-4 significantly enhanced the stimulatory actions of phorbol 12-myristate 13-acetate, ionomycin, and epidermal growth factor but not interleukin-1β on prostaglandin E.sub.2 production. Conclusion: Interleukin-4 stimulates prostaglandin E.sub.2 production by chorion and decidual cells. These data suggest that interleukin-4 production by immune effector cells in gestational tissues may contribute to the pathophysiologic features of preterm **labor**

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CT. . . growth factor; interleukin 1beta; ionomycin; phorbol 13 acetate 12 myristate; article; cell culture; controlled study; human; human cell; placenta; premature **labor**; priority journal; radioimmunoassay; etiology

RN (prostaglandin e2) 363-24-6; (epidermal growth factor) 62229-50-9; (ionomycin) 56092-81-0; (phorbol 13 acetate 12 myristate) **16561-29-8**

AN 1998:226095 BIOSIS
DN PREV199800226095
TI Involvement of phosphatidate phosphohydrolase in arachidonic acid mobilization in human amnionic WISH cells.
AU Balboa, Maria A.; Balsinde, Jesus; Dennis, Edward A. [Reprint author]
CS Dep. Chem. Biochemistry, Sch. Med., Univ. California San Diego, La Jolla, CA 92093-0601, USA
SO Journal of Biological Chemistry, (March 27, 1998) Vol. 273, No. 13, pp. 7684-7690. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 20 May 1998
Last Updated on STN: 20 May 1998
AB Prostaglandins are known to play a central role in the initiation of **labor** in humans, and amnionic cells constitute a major source of these compounds. Prostaglandin synthesis and release by amnion cells in response to hormones and ligands takes place after a characteristic 4-5 h lag. However, we report herein that free arachidonic acid (AA), the metabolic precursor of prostaglandins, can be induced at much shorter times (1 h) in human amnionic WISH cells by phorbol 12-myristate 13-acetate (PMA) through activation of protein kinase Calpha (PKCalpha). WISH cells were found to possess both cytosolic group IV phospholipase A2 (cPLA2) and Group VI Ca2+-independent phospholipase A. (iPLA2). Of these, the cPLA2 was found to be the likely mediator of AA mobilization in PMA-activated WISH cells. PMA also activates phospholipase D (PLD) in these cells and ethanol, a compound that inhibits PLD-mediated phosphatidic acid (PA) formation, blocked AA release. Moreover, prevention of PA dephosphorylation by the PA phosphohydrolase inhibitors propranolol and bromoenol lactone, resulted in inhibition of AA release by PMA-treated-WISH cells. Collectively, these data suggest that activation of cPLA2 and attendant AA release by phorbol esters in WISH cells requires prior generation of DAG by phosphatidate phosphohydrolase.
AB Prostaglandins are known to play a central role in the initiation of **labor** in humans, and amnionic cells constitute a major source of these compounds. Prostaglandin synthesis and release by amnion cells in.

RN 506-32-1 (arachidonic acid)
16561-29-8 (phorbol 12-myristate 13-acetate)
9025-77-8 (phosphatidate phosphohydrolase)
9001-84-7 (phospholipase A)
9001-87-0 (phospholipase D)
141436-78-4 (PROTEIN KINASE C)

ACCESSION NUMBER: 1997:457489 HCAPLUS
DOCUMENT NUMBER: 127:146000
TITLE: An analysis of the mechanisms involved in the **okadaic acid**-induced contraction of the estrogen-primed rat uterus

AUTHOR(S): Arteche, Elena; Strippoli, Giuseppe; Loirand, Gervaise; Pacaud, Pierre; Candenas, Luz; Molto, Juan-Carlos; Souto, Luisa; Fernandez, Javier; Norte, Manuel; Martin, Julio D.; Jean-Pierre, Savineau

CORPORATE SOURCE: Dep. of Physiology, University of Bordeaux II, Bordeaux, 33076, Fr.

SOURCE: Journal of Pharmacology and Experimental Therapeutics (**1997**), 282(1), 201-207
CODEN: JPETAB; ISSN: 0022-3565

PUBLISHER: Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The contractile effect of **okadaic acid** (OA) and its derivs. was investigated in the rat uterus. OA (20 μM) induced a transient contraction which, after plateauing, slowly decreased. The structurally related compound okadanol (20 μM) failed to induce any significant contraction. Conversely, the synthetic compound Me okadaate (20 μM) and the naturally occurring ester 7'-hyroxy-4'-methyl-2'-methylenehept-4'(E)-enyl okadaate (20 μM) were as active as the free acid. The OA-induced contraction was unaffected in the presence of neomycin (5 mM), mepacrine (30 μM), 1-[N,O-bis(1,5-isoquinolinesulfonyl)-N-methyl-L-tyrosyl]-4-phenylpiperazine (10 μM), calphostin C (3 μM) and 1-(5-isoquinolinylsulfonyl)-2-methylpiperazine (30 μM). The **calmodulin** inhibitor N-(6-aminohexyl)-5-chloro-1-naphthalenesulfonamide hydrochloride (100 μM) did not modify the amplitude of the OA-induced contraction but significantly increased the rate of tension decay. The myosin light chain kinase inhibitor 1-(5-chloronaphthalene-1-sulfonyl)-1H-hexahydro-1,4-diazepine hydrochloride (1 mM) significantly reduced the peak amplitude of the contraction. Staurosporine (0.03-0.1 μM) did not modify the contractile component of the OA-induced response but inhibited the subsequent decrease in tension. In freshly dispersed myometrial cells loaded with the fluorescent Ca++ indicator indo 1, OA did not produce any significant increase in [Ca++]i. OA (5- to 90-min contact) also failed to modify the intracellular levels of arachidonic acid, compared with basal values. These data suggest that in the rat **uterus** (1) the **contractile** effect of OA (20 μM) is specifically mediated by inhibition of protein phosphatases type 1 and/or 2A and is related to a direct interaction with the contractile machinery; (2) the decreasing phase of the OA-induced mech. response could be mediated by a staurosporine-sensitive protein kinase different from protein kinase C.

AN 2003-24475 DRUGU P
TI MEK inhibitor **U0126** delays RU486-induced preterm **labor** in rats.
AU Li Y; Je H; Morgan K G; Malek S
CS Harvard-Med.Sch.; Univ.Boston
LO Boston; Watertown, Mass., USA
SO Anesthesiology (98, Suppl. 1, 11, 2003) 4 Ref.
CODEN: ANESAV ISSN: 0003-3022
AV Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, Massachusetts, U.S.A.
LA English
DT Journal
FA AB; LA; CT
FS Literature
AN 2003-24475 DRUGU P
AB The effect of the MEK activation inhibitor U-0126 on RU-486-induced pre-term **labor** was investigated in Sprague-Dawley rats. 18-Day pregnant rats were pretreated with U-0126 (100 mg/kg/6 hr, s.c.) and **labor** was induced on day 19 with RU-486 (2 mg/kg, s.c.). Treatment with U-0126 delayed the onset of parturition to an average of 25.18 hr after RU-486. Delayed **labor** was associated with activation of ERK2 and phosphorylation of caldesmon in myometrium compared to the sham group.Results suggest that the ERK/caldesmon pathway might be used a target for the development of tocolytics. (conference abstract: Society for Obstetric Anesthesia and Perinatology 35th Annual Meeting, Phoenix, Arizona, USA, May 14-17, 2003). (No EX).
ABEX (E97)
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CT [01] U-0126 *PH; **LABOR** *OC; DR9800013 *RN; IN-VIVO *FT; RAT *FT; S.C. *FT; TOCOLYTIC *FT; CALDESMON *FT; EC-2.7.1.37 *FT; LAB.ANIMAL *FT; INJECTION *FT; PROTEIN-KINASE. . .